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High risk and low prevalence diseases: Spontaneous cervical artery dissection

Brit Long [a,*,1], Jessica Pelletier [b], Alex Koyfman [c], Rachel E. Bridwell [d]

[a] Department of Emergency Medicine, Brooke Army Medical Center, Fort Sam Houston, TX, USA
[b] Department of Emergency Medicine, Washington University School of Medicine, St. Louis, MO, USA
[c] Department of Emergency Medicine, UT Southwestern, Dallas, TX, USA
[d] Department of Emergency Medicine, Madigan Army Medical Center, Tacoma, WA, USA

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ABSTRACT

Introduction: Spontaneous cervical artery dissection (sCAD) is a serious condition that carries with it a high rate of morbidity and mortality.
Objective: This review highlights the pearls and pitfalls of sCAD, including presentation, diagnosis, and management in the emergency department (ED) based on current evidence.
Discussion: sCAD is a condition affecting the carotid or vertebral arteries and occurs as a result of injury and compromise to the arterial wall layers. The dissection most commonly affects the extracranial vessels but may extend intracranially, resulting in subarachnoid hemorrhage. Patients typically present with symptoms due to compression of local structures, and the presentation depends on the vessel affected. The most common symptom is headache and/or neck pain. Signs and symptoms of ischemia may occur, including transient ischemic attack and stroke. There are a variety of risk factors for sCAD, including underlying connective tissue or vascular disorders, and there may be an inciting event involving minimal trauma to the head or neck. Diagnosis includes imaging, most commonly computed tomography angiography of the head and neck. Ultrasound can diagnose sCAD but should not be used to exclude the condition. Treatment includes specialist consultation (neurology and vascular specialist), consideration of thrombolysis in appropriate patients, symptomatic management, and administration of antithrombotic medications.
Conclusions: An understanding of sCAD can assist emergency clinicians in diagnosing and managing this potentially deadly disease.

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1. Introduction

This article series addresses high risk and low prevalence diseases that are encountered in the emergency department (ED). Much of the primary literature evaluating these conditions is not emergency medicine focused. By their very nature, many of these disease states and clinical presentations have little useful evidence available to guide the emergency physician in diagnosis and management. The format of each article defines the disease or clinical presentation to be reviewed, provides an overview of the extent of what we currently understand, and finally discusses pearls and pitfalls using a question and answer format. This article will discuss spontaneous cervical artery dissection (sCAD). This condition's low prevalence but high morbidity and mortality, as well as its variable atypical patient presentations and challenging diagnosis, make it a high risk and low prevalence disease. Of note, this review will not discuss in detail cervical artery dissection due to blunt or penetrating injury.

1.1. Definition

sCAD involves either the carotid artery or vertebral artery and occurs as a result of compromise of the arterial wall layers. This can lead to luminal stenosis, which may result in cerebral hypoperfusion and thromboembolic complications [1-6]. This is a common cause of stroke in younger patients [4-9]. While uncommon, sCAD is associated with significant morbidity and even mortality [4,6,9]. sCAD can be divided into the vessel affected (internal carotid versus vertebral) and location (extracranial versus intracranial) [4,6,9].

1.2. Pathophysiology

A dissection is a tear or separation of arterial wall layers, resulting in a false lumen where blood may enter the vessel wall between the intima

* Corresponding author.
E-mail address: Brit.long@yahoo.com (B. Long).
[1] Present Address: 3551 Roger Brooke Dr, Fort Sam Houston, TX 78234, Unites States.

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and the media or between the media and the adventitia [1,3-6]. Hemorrhage can occur due to intimal tear or be due to rupture of the vessel wall. Dissections affecting the subintimal area can result in luminal stenosis and complete occlusion, while subadventitial dissections can cause formation of a dissecting aneurysm [1,3-6]. A false lumen may extend back into the true lumen, resulting in a double channel for blood flow [3,4]. Intramural hemorrhage, subintimal plane destruction, and separation of the media and adventitia may be present [1,3-7].

An intramural hematoma and subintimal dissection can cause luminal stenosis and occlusion, leading to cerebral ischemia from hypoperfusion, thromboembolism, or a combination of both. Current literature suggests that thromboembolism is the most likely underlying cause of cerebral ischemia, rather than hypoperfusion, in the setting of sCAD [6,8-13]. Subadventitial dissections that result in formation of an aneurysm or hematoma, and vessel dilation can result in compression of local structures such as nerves and other vasculature [4,6].

The mechanism of sCAD is not completely understood and is likely multifactorial, occurring due to a combination of genetic, environmental, and acquired risk factors [4,6]. While most cases occur with a mechanical trigger, many patients will not recall an inciting event and are unaware of any major risk factors that lead to the vascular injury and dissection [14]. Risk factors include connective tissue disorders (e.g., fibromuscular dysplasia, Ehlers-Danlos syndrome) and vascular disease [13,15]. Ultimately, sCAD may lead to compressive symptoms due to involvement of structures around the dissection and ischemic symptoms due to vascular occlusion or emboli [3,4].

sCAD affecting the carotid may be intracranial or extracranial (Fig. 1). Intracranial carotid artery dissections most frequently occur in the supraclinoid segment, while extracranial carotid dissections occur 2 cm distal to the carotid bifurcation, around the skull base (Fig. 1) [4,16]. Vertebral dissection most commonly occurs in the transverse processes of C2-C6, affecting the V2 segment, or the C2 transverse process and foramen magnum at the skull base, affecting the V3 segment [16-18]. Importantly, multiple dissections are present in up to 22% of cases, more commonly in females, though <2% have three or more dissections simultaneously [6,18-26]. Dissections involving intracranial arteries are at a higher risk of rupture, as they have only a thin adventitial layer with no external elastic lamina as compared to the more robust extracranial arteries; this may lead to subarachnoid hemorrhage [6,18-26].

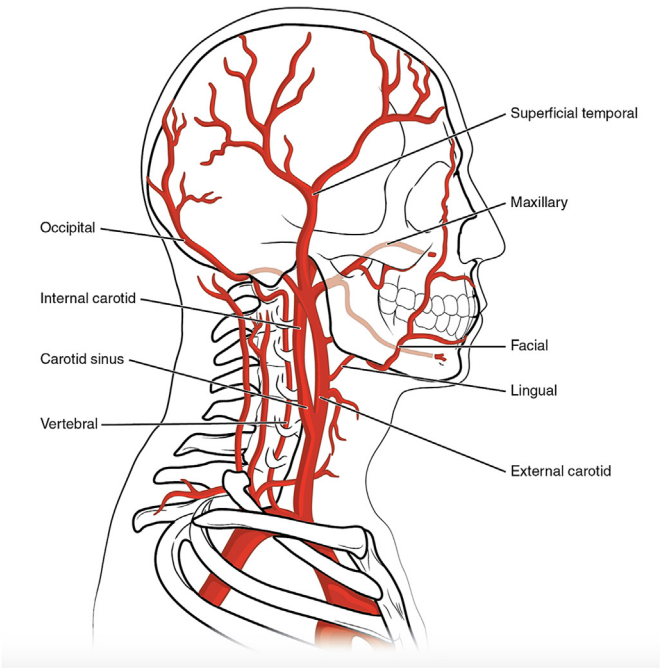

Fig. 1. Arterial vessels of the neck. Obtained from https://commons.wikimedia.org/wiki/File:2122_Common_Carotid_Artery.jpg.

1.3. Epidemiology

sCAD is not common, with one study suggesting an incidence of 1.72 per 100,000 individuals for internal carotid artery dissection, 0.97 per 100,000 individuals for vertebral artery dissection, and 2.6–3 per 100,000 for combined cases [4,6,20-23]. However, this is likely an underestimation, as many patients are never diagnosed. Literature suggests that sCAD accounts for approximately 2% of strokes overall, but in patients <45 years, 15–24% of strokes are due to sCAD [4-7,27]. While this affects a younger population, with a mean age of 35 years to 53 years, there does not appear to be a predilection based on sex or ethnicity [8,12,19,20,22,24,28,29]. The literature is inconsistent regarding differences in incidence based on sex, with studies in North America suggesting women are more commonly affected, but in European studies men are more commonly affected [8,12,19,20,22,24,28,29]. Extracranial dissection is more common than intracranial dissection in North America and Europe, but intracranial dissection is more common in pediatric patients and Asian populations [4,8,20,23,24,29].

2. Discussion

2.1. ED presentation

Patients with sCAD can present with a variety of signs and symptoms. A significant number may present with isolated local symptoms or may even be asymptomatic [12,30-33]. Local symptoms depend on the vessel involved; the most common symptom is ipsilateral head or neck pain, which is often severe and continuous [4,20,30-37]. Pain may be sudden in onset. Patients with carotid artery dissections typically have anterior head and neck pain, whereas those with vertebral artery dissection more commonly have occipital pain and posterior neck pain [4,20,30-34]. Horner syndrome may be observed due to distension of the sympathetic fibers along the external surface of the internal carotid artery [4,20,35-37]. This presentation is typically partial with ptosis and miosis but no anhidrosis [4,20,36,37]. Cranial neuropathies may be present due to compression, most commonly cranial nerve XII followed by IX [18,19,38,39]. Pulsatile tinnitus may be isolated or occur in combination with other symptoms [40].

Dissection can lead to transient ischemic attack (TIA) or stroke and should be suspected when neurologic findings are present in the setting of neck pain and/or headache [18,20,24,32]. The risk of stroke appears to be the highest in the first two weeks following the dissection [41]. Ischemic syndromes are not necessarily specific to the dissection but are associated with the vascular territory affected by subsequent cerebral ischemia [18,20,24,32,41]. Carotid artery dissections can present with monocular blindness, unilateral extremity weakness, and sensory changes. Vertebral artery dissections can lead to posterior circulation (PC) stroke or cervical spinal infarction [42,43]. PC stroke may present as severe vertigo, ataxia, dysmetria, nausea/vomiting, vision changes, and weakness, and cervical spinal infarction with extremity weakness or sensory changes. Dissection extending intracranially with subarachnoid hemorrhage may present with altered mental status, focal neurologic deficits, and severe headache [4,6,27,30].

2.2. ED evaluation

Laboratory analysis does not assist in the diagnosis of sCAD; imaging is necessary, which typically includes computed tomography angiography (CTA) of the head and neck [4,6,27]. This can both confirm the diagnosis and guide management. CTA may demonstrate stenosis or occlusion, intimal flap, dissecting aneurysm, intramural hematoma, or

a dilation of the involved vessel. Magnetic resonance imaging (MRI) and MR angiography (MRA) may also be used to diagnose sCAD, though these are less often available in the ED setting [44-46]. Carotid duplex and transcranial Doppler ultrasound can be used to monitor therapy but may miss the diagnosis, particularly cases with dissection involving the skull base or within the transverse foramina; thus, these imaging modalities are more relevant for the inpatient management of sCAD [47,48].

2.3. ED management

Management focuses first on evaluating for the effects of acute cerebral ischemia induced by sCAD. For those who present with TIA or acute ischemic stroke in the setting of cervical artery dissection, management includes assessment of the airway and need for intervention, blood pressure regulation, control of any metabolic derangements such as hyperglycemia, and consideration of reperfusion [9,49-55]. Patients with a neurologic deficit who present within 4.5 h of the deficit with no contraindications to thrombolysis and no intracranial involvement may be candidates for thrombolysis (e.g., alteplase or tenecteplase) to restore blood flow and reduce disability [9,54]. The presence of cervical artery dissection alone is not a contraindication to thrombolysis, and mechanical thrombectomy is also an option. Patients who present outside of the thrombolytic window or those with no neurologic deficit may be managed with antithrombotic therapy (i.e., anticoagulation or antiplatelet therapy). Antithrombotic therapy is necessary due to the increased risk of thromboembolic events with sCAD [9,49-55]. The duration of treatment is typically 3–6 months, followed by repeat imaging [4,6,9,20,49-55]. Patients should also receive symptomatic therapy. Neurology and vascular specialist consultation is recommended, depending on the hospital, and patients are typically admitted for further evaluation and management. Consultation with the neurosurgery specialist is necessary if subarachnoid hemorrhage is present.

3. Pearls and pitfalls

3.1. What can lead to sCAD, and what are the major risk factors?

While sCAD contains "spontaneous" in the name, literature suggests that minor cervical trauma precedes up to 41% of spontaneous cases of sCAD [4,6,13,15], though approximately one third will not report a mechanical event [4,6,13,15]. Triggers include overt, major trauma or minor trauma such as chiropractic manipulation, minor sport injuries, painting a ceiling, dancing with head movements, intense cough/sneezing/vomiting, childbirth, valsalva maneuver during childbirth or the postpartum period, neck cracking, whiplash, yoga, scuba diving, weightlifting, trampoline jumping, and roller coasters [4,6,22,56-58].

A variety of risk factors are associated with sCAD. Vascular or connective tissue disorders are the most commonly associated risk factors, but they are not present in the majority of patients. Connective tissue disorders include fibromuscular dysplasia, Ehler-Danlos syndrome type IV, Marfan syndrome, osteogenesis imperfecta, and polycystic kidney disease [4,6,9,13,29,59-63]. The most common is fibromuscular dysplasia, accounting for 15–40% of cases [59]. Ehlers-Danlos syndrome type IV accounts for <2% [60,62]. Other associated factors include recent infection, head or neck surgery, cardiovascular risk factors, migraine, smoking, oral contraceptive use, hypertension, elongated styloid process, and pregnancy [22,64,65]. Ultimately, the emergency clinician should consider these risk factors in the history, but the absence of these factors should not be used to exclude a diagnosis of sCAD.

3.2. What history and examination findings suggest the disease?

Patients most commonly present with neck pain and/or headache, which are nonspecific. Other findings may be subtle, and thus the diagnosis is often missed on initial presentation, with a delay in diagnosis of approximately 9 days [4,6,66]. Most signs and symptoms are due to pain, compression of surrounding structures from the vessel, ischemia from embolus or vascular occlusion, or even subarachnoid hemorrhage. Pain is the most common symptom, typically in the head, face, and/or neck in 57–80% of patients [3-34,67]. Headache is present in up to 68% of patients [33,67]. The headache may be sudden and maximal in onset but is more commonly described as gradually worsening, severe, unilateral (ipsilateral to the dissection), and continuous in the frontal or temporal regions for carotid artery dissection and in the posterior regions for vertebral artery dissections [20,30-34,67]. Pain in the face, ear, eye, and/or teeth occurs in 34–53% of patients, which is more common in carotid artery dissection. Literature suggests neck pain occurs in 9–46% of cases overall and is more common in those with vertebral artery dissection [32,33,67]. Neck pain occurs in up to 46% of those with vertebral artery dissection and is typically posterolateral in location [32,67]. Approximately 18% of those with carotid artery dissection will have neck pain, typically over the anterolateral neck with radiation to the jaw or ear. A bruit may occur in 33% of cases and is more common in vertebral artery dissections [4,6,33,67]. Of note, some form of head or neck pain may be the only symptom in 8% of cases [40].

In addition to head and neck pain, focal neurologic deficits may also be present in sCAD. Neurologic compression may result in cranial nerve palsies in up to 16% of patients, most commonly cranial nerve XII, followed by IX, X, and XI in carotid artery dissection [4,6,18,33,39,67,68]. Partial Horner syndrome with miosis and ptosis but no anhidrosis occurs in 25–40% of carotid dissections as a result of artery expansion and sympathetic fiber compression within the internal carotid artery plexus [20,33,66,69]. Vertebral artery dissections associated with brainstem stroke affecting the central sympathetic fibers may result in Horner syndrome, though this is rare. Involvement of the glossopharyngeal nerve or chorda tympani may result in dysgeusia in 7% of patients, which is specific for carotid artery dissection [4,6,27]. Vertebral artery dissection with expansion of the external layers or aneurysm formation can compress the cervical nerve roots, leading to radiculopathy. The most common nerves affected are C5 and C6, resulting in weakness of the biceps, triceps, deltoid, infraspinatus, supraspinatus, and rhomboid, as well as altered sensation in the lateral arm and hand [70,71]. Decreased or absent biceps and brachioradialis reflexes may be found with C5/C6 involvement. Pulsatile tinnitus occurs in 8–27% of carotid artery dissections due to transmission of the sound of non-laminar blood flow in the narrowed blood vessels to the inner ear [33,67,72]. Importantly, the classic triad of carotid artery dissection including headache, partial Horner syndrome, and contralateral stroke symptoms is present in <8% of cases [73].

Ischemic signs and symptoms typically follow the compressive symptoms, though patients may develop evidence of ischemia without compressive symptoms. These include TIA or stroke, which affect up to 67% of patients with cervical vessel dissection, with 23% experiencing TIA and 56% stroke [12,20,33,74]. Ischemia typically occurs within the first week of compressive symptoms and is more common in younger patients with an average age of 45 [32,74]. Compared to young patients with strokes from other etiologies, patients with cervical vessel dissection resulting in stroke are less likely to have cardiovascular disease, hypertension, hyperlipidemia, a patent foramen ovale, obesity, or tobacco dependence [4,27,75]. Overall, the most common presenting signs and symptoms for stroke associated with dissection include vertigo, nausea, vomiting, and vision changes, and patients more frequently report headaches compared to those with strokes from other etiologies [4,27,75]. Carotid artery dissection resulting in ischemia affects the middle cerebral and anterior cerebral arteries, resulting in signs and symptoms such as hemisensory changes, hemiparesis, gaze deviation, hemineglect, dysarthria, aphasia, and/or vision changes [4,27,32]. Vertebral artery dissections affect the posterior circulation and result in vertigo, ataxia, dysmetria, Horner syndrome, hemiplegia, nausea/vomiting, nystagmus, diplopia, dysarthria, and/or dysphagia [4,27,32]. Spinal cord infarction may occur in the setting of vertebral artery

dissection, though this is rare [4,27]. Subarachnoid hemorrhage occurs in 8–69% of those with intracranial artery dissection [8]. Ultimately, the disease should be considered in patients with headache, neck pain, and/or ischemic symptoms in several situations, detailed in Table 1.

3.3. What are dangerous mimics of sCAD?

Due to the nonspecific presentation of sCAD, there are a variety of mimics. Dangerous mimics include subarachnoid hemorrhage, intracerebral hemorrhage, reversible cerebral vasoconstriction syndrome, cerebral infection (e.g., meningitis, abscess), cerebral venous thrombosis, hypertensive encephalopathy, posterior reversible leukoencephalopathy syndrome, pituitary apoplexy, giant cell arteritis, and acute angle closure glaucoma, among others [76]. Table 2 provides a summary of these conditions.

3.4. What imaging is recommended?

A variety of imaging tests may be used for diagnosis. The most readily available test in the ED is CTA of the neck and head (Figs. 2 and 3). This imaging modality demonstrates a significant range of sensitivities, from 50% to 100% with specificity 90–100% [4,27,42-44,77-80]. CTA may demonstrate a thrombus, focal stenosis and distal dilation (string and pearl sign), intimal flap, double lumen, luminal occlusion, or pseudoaneurysm [4,27,42-44,77-80]. Of note, CTA is preferred for the diagnosis of vertebral artery dissections, and more recent studies published in the last 5 years demonstrate sensitivities and specificities >90% for dissection involving the internal carotid or vertebral artery [4,6,27].

MRI and MRA can be used for diagnosis and evaluation of sCAD and subsequent ischemic stroke, though lack of availability in many centers and imaging acquisition time serve as potential barriers (Fig. 4) [42,44-46,79-82]. If acquired, findings may include intramural hematoma, intimal flap, double lumen, vessel dilation, and pseudoaneurysm [4,27,80]. An intramural hematoma is the most reliable finding on MRI, which appears as a crescent hyperintense rim on T1-weighted imaging in the initial stages; this progresses to a hyperintense signal on T1 and T2 weighted images over 1 week [42,44-46,79-82]. MRI with MRA has a sensitivity of 83–99% and specificity of 99% for dissections involving the carotid artery, but the sensitivity ranges between 20 and 60% for vertebral artery dissections [4,27,80]. Of note, MRI and MRA demonstrate higher sensitivity for the diagnosis of ischemic stroke [4,27,80].

Ultrasound can directly visualize the dissection/intimal flap, detect intramural hematoma, and evaluate vascular flow in real time [47,48,83-86]. However, there are several limitations including a sensitivity ranging between 38 and 86% [47,48,83-86]. Ultrasound is unable to visualize the vasculature around the base of the skull and the majority of the vertebral artery through the transverse foramina. Thus, this modality should not be used to exclude the diagnosis of sCAD but can be used to rule it in, as well as in evaluating the progression of a known sCAD with serial ultrasound evaluations.

Digital subtraction angiography (DSA) is classically considered the "gold standard" for diagnosis sCAD but is unnecessary if CTA or MRI/MRA demonstrates dissection [4,27,80]. Angiography does provide

Table 1
Scenarios to consider sCAD.

1) Headache, neck pain, and/or stroke symptoms AND the following:
2) Neurologic symptoms concerning for ischemic stroke, Horner syndrome, pulsatile tinnitus, cranial/cervical neuropathies, tooth/eye pain without apparent pathology OR
3) Recent inciting events (e.g., minor or trivial trauma, sporting events or activity, sneezing/coughing) OR
4) Personal or family history of connective tissue disorder, vascular disorder, or migraine

Table 2
Severe mimics of sCAD.

Diagnosis	Features
Subarachnoid hemorrhage	Sudden, severe headache; may have had sentinel headache in the past Neck pain, neck stiffness on examination Transient or continued alteration or loss of consciousness Focal neurologic deficit
Reversible cerebral vasoconstriction syndrome	Multiple presentations with sudden severe headache Headache only lasts several hours but will reoccur
Cerebral venous thrombosis	Postpartum, recent surgery or infection, or hypercoagulable state Headache, papilledema, focal neurologic deficit, seizures, altered mental status Patients often younger than 50 years LP with elevated opening pressure
Posterior reversible encephalopathy syndrome	Headache, seizures, visual changes; extreme hypertension Most patients will be altered
Spontaneous intracranial hypotension	Recent spinal procedure, positional headache that worsens when upright
Pituitary apoplexy	History of pituitary adenoma, ophthalmoplegia, vomiting, decreased visual fields
Third ventricle colloid cyst	Headache that is sudden but then rapidly resolves; signs of hydrocephalus
Acute angle closure glaucoma	Eye pain and referred headache, vision changes, nausea/vomiting Cornea is injected and hazy, midrange fixed pupil Globe is hard to palpation; intraocular pressure > 20 mmHg
Meningitis	Fever, headache, meningismus, change in mental status LP demonstrates elevated white blood cells with neutrophil predominance, low CSF glucose/elevated protein, positive PCR/culture, elevated CSF lactate

Abbreviations: CSF – cerebrospinal fluid, LP – lumbar puncture, PCR – polymerase chain reaction, TCH – thunderclap headache.

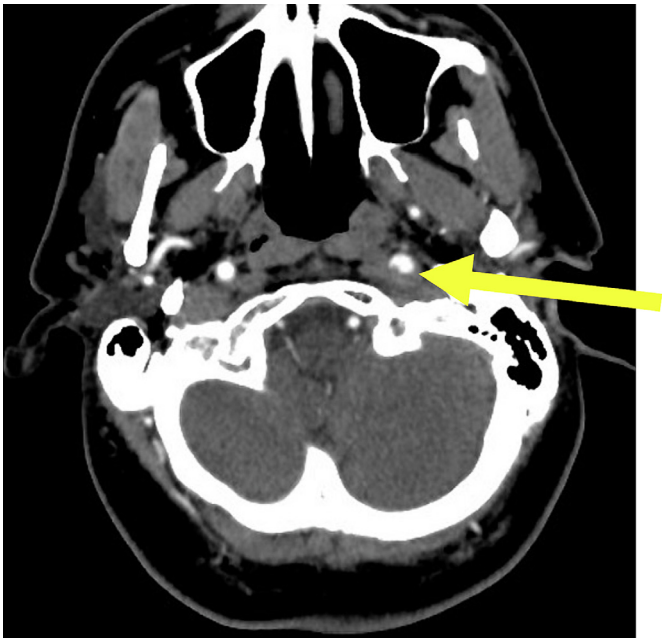

Fig. 2. CTA with left internal carotid artery demonstrating an abnormal vessel contour, with mildly narrowed distal segment surrounded by a non-enhancing crescent-shaped mural thrombus (yellow arrow). Case courtesy of Andrew Dixon, Radiopaedia.org, rID: 53541.

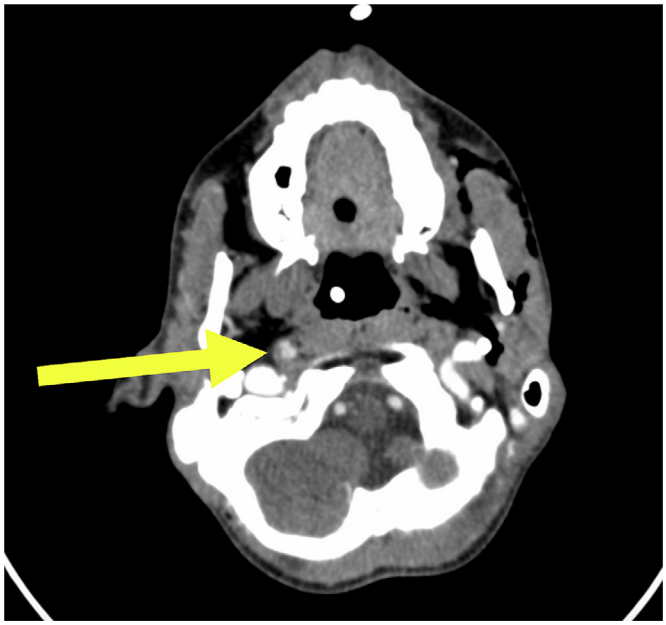

Fig. 3. CTA demonstrating crescent-shaped hyperattenuating focus on the right internal carotid artery (ICA), representing intramural hematomas, with an intimal flap and double-lumen (yellow arrow). Case courtesy of Antonio Rodrigues de Aguiar Neto, Radiopaedia.org, rID: 96007.

assessment of vascular flow (which is not possible with CTA and MRI/MRA), but it is labor intensive and is associated with risks including stroke, bleeding, and pseudoaneurysm formation [4,27,80]. If initial imaging is unremarkable, DSA should be pursued in conjunction with specialist consultation [4,27,80].

3.5. What should be considered for management?

The management of confirmed sCAD first focuses on whether the patient has stroke signs and symptoms with neurologic deficits in combination with neurology and vascular surgery consultation [4,6,9,20,49-55]. If intracranial dissection or subarachnoid hemorrhage is present, consultation with the neurosurgery specialist is recommended. Other pertinent considerations include the presence of a pseudoaneurysm and hemorrhage. If the patient has evidence of ischemic stroke, sCAD is not a contraindication to treatment with thrombolytics (e.g., alteplase, tenecteplase), with management based on local acute stroke protocols [4,6]. A 2009 retrospective study evaluating thrombolytics in sCAD found no difference in rates of intracranial hemorrhage or recurrent ischemic stroke compared with non-sCAD patients [4,6,50-55]. Another retrospective study found no difference in modified Rankin scale (mRS) or adverse events between sCAD patients receiving thrombolytics and controls with non-sCAD ischemic stroke [54]. Patients who present within 4.5 h of neurologic deficit, no intracerebral hemorrhage, and no involvement of the aorta or intracranial vasculature are candidates for thrombolysis [9]. There are no randomized controlled trials evaluating the use of thrombolytics in those with cervical vessel dissection and ischemic stroke, though initial trials evaluating thrombolysis did not exclude those with sCAD [4,87]. However, safety data regarding the use of IV thrombolysis in those with intracranial dissection are lacking [88]. The CADISP study evaluated 3-month outcomes and rates of major bleeding and found that of the 618 sCAD patients presenting with ischemic stroke, 11% received thrombolytics [32]. These patients had a National Institutes of Health Stroke Scale [NIHSS] score of 16 compared to 3 for those not receiving thrombolytics. There was no difference in neurologic outcomes between groups, but there was an increase in hemorrhage with thrombolytics of 5.9% versus 0.6% in those who do not receive thrombolytics [32].

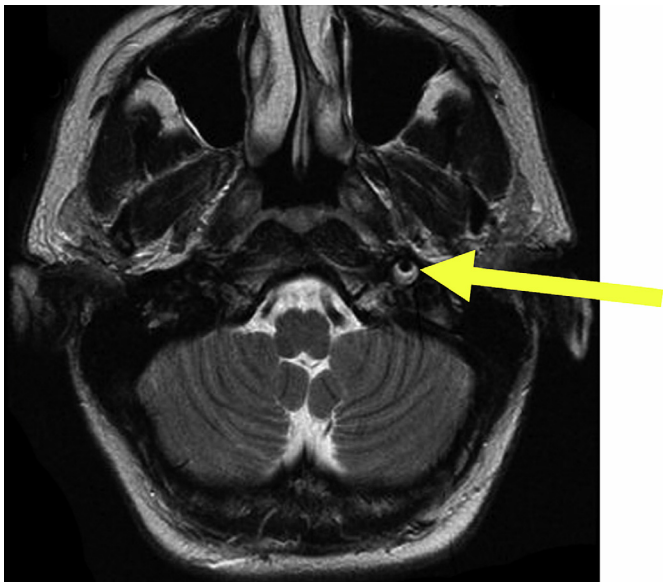

Fig. 4. MRI with T2-weighted MRI showing blood in the arterial wall and narrowing of the lumen of the left internal carotid artery (yellow arrow), known as the "crescent sign." Case courtesy of Frank Gaillard, Radiopaedia.org, rID: 6693.

Prevention of ischemic stroke with antithrombotic medication is integral, with a focus on reducing the risk of artery-to-artery embolic stroke. Antithrombotic medication reduces the risk of stroke from approximately 26–57% to 3–4% and includes either anticoagulation (e.g., heparin with transition to other measures such as warfarin) or antiplatelet medication (e.g., aspirin or clopidogrel) [4,6,9,49-55]. The specific choice of agent is controversial, with several studies finding similarly low rates of stroke for patients receiving antiplatelet versus anticoagulant medications [9,49,55,89].

Ultimately, the decision to initiate anticoagulation versus antiplatelet agent should be made with specialist consultation. The 2021 AHA/ASA guidelines recommend either warfarin or aspirin in patients with recent TIA or ischemic stroke associated with extracranial sCAD with consideration of concomitant injuries in trauma patients [90]. Importantly, antithrombotic medication should be delayed for 24 h following thrombolysis if administered. Patients with another indication for antiplatelet therapy should likely receive antiplatelet therapy; this is also less expensive, does not require monitoring, and may be associated with lower risk of hemorrhage compared with anticoagulation. Patients with local compressive symptoms but no ischemia, large strokes or intracranial extension, dissections with small intimal tears, or those with <25% luminal narrowing may also benefit from antiplatelet therapy [4,6,9,27]. Anticoagulation may be optimal for patients who have another indication for anticoagulation, and a heparin infusion may be stopped rapidly if there is concern for bleeding [4,9]. Anticoagulation is also likely more effective for those with free-floating thrombi, multiple ischemic events in the same vascular distribution, or severe vascular occlusion or stenosis [9]. While direct oral anticoagulants (DOACs) were not utilized in CADISS or TREAT-CAD, data from observational studies suggest DOACs are effective and safe [9,49,55]. Treatment duration is 3–6 months, but the optimal duration remains unclear [9,90].

Endovascular interventions for sCAD include stenting, angioplasty, or embolization of the dissected vessel [4,6,9,91,92,93]. These are typically pursued for those with contraindications to antithrombotic medications, recurrent strokes despite medical therapy, severe occlusion or vascular narrowing, large vessel occlusion stroke (LVO), severely compromised cerebral blood flow, or a pseudoaneurysm that is enlarging [94,95]. Placement of a stent requires subsequent dual antiplatelet therapy.

3.6. Are there recommendations concerning blood pressure management for sCAD?

There are no clear data concerning optimal blood pressure targets in those with sCAD, though hypertension is a risk factor for sCAD. If the patient presents with ischemic symptoms and is a candidate for thrombolytics, an initial blood pressure target <185/110 mmHg prior to administration of thrombolytics followed by <180/105 mmHg for the first 24 h is recommended [4,6,96]. If subarachnoid hemorrhage occurs, a target systolic blood pressure < 160 mmHg is recommended [4,27,96]. An agent such as nicardipine or clevidipine can provide rapid control of blood pressure. Of note, hypotension should be avoided in order to prevent cerebral hypoperfusion.

3.7. What are considerations for intracranial dissection?

Intracranial dissection can occur as the primary site or from extension of extracranial dissection. While intracranial dissections are less common compared to extracranial dissections, these more commonly lead to ischemic stroke, occurring in up to 30–84% of cases, and subarachnoid hemorrhage, occurring in 15–60% of cases [20,32,97-99]. Subarachnoid hemorrhage is the leading cause of morbidity and mortality in those with intracranial dissection [4,6,9,56]. Antiplatelets are typically recommended over anticoagulants for treatment due to the risk of bleeding. In those with subarachnoid hemorrhage, rebleeding occurs in over 58% of patients and increases the risk of mortality [100,101]. Dissection associated with subarachnoid hemorrhage requires neurosurgery consultation for emergent endovascular or surgical evaluation and repair [4,9,102].

3.8. What are the long-term outcomes for those with sCAD?

Vessel healing begins within the first few months following dissection. Literature suggests that 60–82% will demonstrate complete healing within 12 months [103-105]. The incidence of recurrent ischemia ranges between 0 and 13% at 1 year [12,49,55], though the risk of stroke is highest within the first 2 weeks. Recurrent dissection may occur, with literature inconsistent regarding exact numbers. The recurrence rate ranges between 2% at 3 months to 16% at 1 year, though most events occur within the first month after initial diagnosis [32,106]. Connective tissue disorders are associated with increased risk of recurrent sCAD [4,6,12,32]. Repeat imaging is typically completed at 3–6 months after diagnosis to assess recanalization and assist with determining need for ongoing antithrombotic medication [4,6,27].

Ultimate functional outcomes and mortality depend on the presence or absence of associated ischemic stroke and subarachnoid hemorrhage. The majority of patients with sCAD have favorable outcomes, with 75–82% demonstrating a mRS score of 0 or 1 and mortality rates of <5% [4,12,20]. Unfortunately, up to 40% of sCAD patients report reduced quality of life despite good functional outcomes [107-109]. Finally, a minority of patients develop prolonged headaches following sCAD [31].

Table 3 provides pearls for sCAD.

4. Conclusion

sCAD affects the carotid or vertebral arteries and may be extracranial and/or intracranial. There are many risk factors, including connective tissue and vascular disorders as well as minor trauma. Signs and symptoms are due to compression of local structures as well as ischemia. The most common symptoms are headache and neck pain, though TIA and stroke may also occur. Intracranial dissection can result in subarachnoid hemorrhage. CTA of the head and neck is the most readily available imaging modality for diagnosis in the ED setting. Treatment includes specialist consultation and antithrombotic medication, though thrombolysis may be considered in select patients presenting with ischemic stroke.

Table 3
Cervical vessel dissection pearls.

- sCAD is divided into carotid versus vertebral, as well as extracranial versus intracranial. The presentation is based on the affected vessel.
- sCAD has multiple risk factors, including connective tissue disorders, and many patients will have a mechanical trigger.
- The most common presentation is head, neck, or face pain. Other signs and symptoms occur due to compression of local structures and ischemia.
- The clinician should consider the diagnosis in those with new headache and/or neck pain, whether or not neurologic symptoms are present.
- A variety of conditions may mimic sCAD, including subarachnoid hemorrhage, reversible cerebral vasoconstriction syndrome, cerebral venous thrombosis, posterior reversible encephalopathy syndrome, spontaneous intracranial hypotension, pituitary apoplexy, third ventricle colloid cyst, acute angle closure glaucoma, and meningitis.
- Imaging may include CTA of the head and neck or MRA. Ultrasound should not be used to exclude the diagnosis.
- Treatment includes antiplatelet or anticoagulant medications to reduce the risk of stroke or recurrent stroke, though some patients may be appropriate for thrombolysis.

CRediT authorship contribution statement

Brit Long: Writing – review & editing, Writing – original draft, Visualization, Validation, Resources, Formal analysis, Conceptualization. **Jessica Pelletier:** Writing – review & editing, Writing – original draft, Visualization, Validation. **Alex Koyfman:** Writing – review & editing, Visualization, Validation, Supervision, Resources, Conceptualization. **Rachel E. Bridwell:** Writing – review & editing, Writing – original draft, Visualization, Resources.

Declaration of Competing Interest

None.

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